IN THE SPECIFICATION

Please amend the specification as shown:

Please delete the paragraph on page 17, lines ~~15-20~~ 11-16, and replace it with the following paragraph:

Figure 35 shows the proliferative response of responder T cells (CAP-M8 T-cell line specific for CEA peptide 8) at various APC ratios harvested on day 5 after stimulation with peptide-pulsed DCs infected with rV-TRICOM and rested 2 days with 10 u/ml IL2 (no APC or peptide). Peptide 8 - (EAQNTTYL) **(SEQ ID NO: 37)** in assay at 1 ug/ml final concentration. ^{3}H-thymidine added on day 2, T cells harvested on day 3. 0=DC(v-Wyeth) - pep and Δ = DC (rV-TRICOM) - pep results are at baseline.

Please delete the Table on page 35, and replace it with the following Table:

Table 1
Antigens and Epitopes Recognized by T Cells

Target antigens	Restriction element	Immunological Peptide epitope	SEQ. ID No.
Human target tumor antigens recognized by T cells			
gp 100	HLA-A2	KTWGQYWZY	1
	HLA-A2	ITDQVPPSV	2
	HLA-A2	YLEPGPVTA	3
	HLA-A2	LLDGTATLRL	4
	HLA-A2	VLYRYGSFSV	5
MART1-/Melan A	HLA-A2	AAGIGILTV	6
	HLA-A2	ILTVILGVL	7
TRP-1 (GP75)	HLA-A31	MSLQRQFLR	8
Tyrosinase	HLA-A2	MLLAVLYCL	9
	HLA-A2	YMNGTMSQV	10
	HLA-B44	SEIWRDIDF	11
	HLA-A24	AFLPWHRLF	12
	HLA-DR4	QNILLSNAPLGPQFP	13
	HLA-DR4	SYLQDSDPDSFQD	14
MAGE-1	HLA-A1	EADPTGHSY	15
	HLA-Cw16	SAYGEPRKL	16
MAGE-3	HLA-A1	EVDPIGHLY	17
	HLA-A2	FLWGPRALV	18
BAGE	HLA-Cw16	AARAVFLAL	19
GAGE-1,2	HLA-Cw6	YRPRPRRY	20
N-acetylglucos-aminyltransferase-V	HLA-A2	VLPDVFIRC	21
p15	HLA-A24	AYGLDFYIL	22
CEA		YLSGANLNL(CAP1)	23

		YLSGADLNL (CAP1-6D)	24 37
β-catenin	HLA-A24	SYLDSGIHF	25
MUM-1	HLA-B44	EEKLIVVLF	26
CDK4	HLA-A2	ACDPHSGHFV	27
HER-2/neu	HLA-A2	IISAVVGIL	28
(Breast and ovarian carcinoma)	HLA-A2	KIFGSLAFL	29
Human papillomavirus-E6,E7 (cervical carcinoma)	HLA-A2	YMLDLQPETT	30
MUC-1	Non-MHC restricted MHC restricted	PDTRPAPGSTAPPAHGVTSA (and portions thereof)	31
(Breast, ovarian and pancreatic carcinoma)			
PSA	A2, A3	FLTPKKLQCVDLHVISNDVCA-QVHPQKVTK	32
		FLTPKKLQCV	33
		KLQCVDLHV	34
		VISNDVCAQV	35
		QVHPQKVTK	36

Please delete the paragraph on page 80, lines 7-12, and replace it with the following paragraph:

Figure 35 demonstrates that DCs infected with rV-TRICOM are far superior than standard DCs in stimulating a CEA peptide-specific murine T cell line. This T-cell line is $CD8^+$ and is specific for the CEA D^b Class-I restricted epitope EAQNTTYL **(SEQ ID NO: 37)** (CAP-M8). The combination of DCs pulsed with the CEA peptide (1μg/ml) and previously infected with rV-TRICOM is clearly superior in stimulating CEA-specific T cell responses, especially at low T-cell to DC ratios.

Please delete the paragraph on page 80, lines 18-23, and replace it with the following paragraph:

The H-$2k^b$-restricted peptides OVA (ovalbumin$_{257-264}$, SIINFEKL)[41] **(SEQ ID NO: 38)** and VSVN (vesicular stomatitis virus N_{52-59}, RGYVYQGL)[42] **(SEQ ID NO: 39)** and the H-$2D^b$ restricted peptides CAP-M8 ($CEA_{526-533}$, EAQNTTYL) **(SEQ ID NO: 37)** and FLU-NP ($NP_{366-374}$, ASNENMDAM)[43] **(SEQ ID NO: 40)** were either purchased (Multiple

Peptide Systems, San Diego, CA) or synthesized in-house (Applied Biosystems 432A Synergy Peptide Synthesizer, Foster City, CA).

Please delete the paragraph on page 89, lines 7-27, and replace it with the following paragraph:

Studies were undertaken to determine if the stimulatory capacity of peptide-pulsed DC could be enhanced by infecting DC with rV-TRICOM. To that end, the H-2K^b-restricted OVA (ovalburnin$_{257-264}$, SIINFEKL) **(SEQ ID NO: 38)** peptide and an OVA-specific CD8$^+$ effector T-cell line were used. DC were exposed to different concentrations of OVA peptide and incubated in the presence of the OVA T-cell line (Fig. 38A-38F). The conventional (i.e., uninfected) DC induced a strong proliferation of OVA-specific T cells when incubated with the OVA peptide (Fig. 38A). These DC did not induce proliferation of OVA-specific T cells when incubated with the control peptide VSVN (vesicular stomatitis virus N$_{52-59}$ RGYVYQGL) **(SEQ ID NO: 39)** (Fig. 38A, open squares). DC/rV-B7-1 increased the overall peptide-specific proliferation of these cells 1.8-fold (Fig. 38C). In addition, DC/rV-B7-1 induced similar proliferation to that of uninfected or mock-infected DC in the presence of 4-fold less peptide. In contrast, DC/rV-TRICOM increased the overall proliferation of these T-cells several-fold, and in the presence of 32-fold less OVA peptide, induced proliferation comparable to that of uninfected DC (Fig. 38C). To further evaluate the capacity of vaccinia-infected DC to present peptide, DC were pulsed with a single concentration of OVA peptide (1 μM) and incubated in the presence of several ratios of T cells (Fig. 38E). On a per-cell basis, 4-fold fewer DC/rV-B7-1 were required to induce proliferation levels comparable to that of DC (open triangles vs. closed squares). The greatest stimulatory effect was that of DC/rV-TRICOM, which induced proliferation levels comparable to that of DC with 32-fold less cells (open circles vs. closed squares).

Please delete the paragraph on page 89, line 28, through page 90, line 2, and replace it with the following paragraph:

A second peptide system employing peptide-pulsed DC and an established T-cell line were employed to determine if results similar to those obtained with the OVA peptide could be noted. These experiments were conducted using the H-2D^b-restricted peptide CAP-M8 ($CEA_{526-533}$, EAQNTTYL) **(SEQ ID NO: 37)** and a CAP-M8-specific $CD8^+$ effector T-cell line; similar results were noted (Fig. 38B, D, F). These experiments were repeated 5 additional times with the same results.

Please delete the paragraph on page 99, line 23, through page 100, line 8, and replace it with the following paragraph:

Peripheral blood lymphocytes and dendritic cells are obtained from a patient with advanced prostate cancer. The peripheral blood lymphocytes are enriched for CD8+ lymphocytes. The dendritic cells are infected with rV-PSA epitope QVHPQKVTK **(residues 22-30 of SEQ ID NO: 32)**/B7.1/ICAM-1/LFA-3 for a period of time sufficient to allow expression of the PSA epitope and overexpression of the multiple costimulatory molecules. PSA epitope-specific $CD8^+$ lymphocytes are activated and expanded in the presence of these treated dendritic cells. The activated PSA epitope-specific $CD8^+$ autologous T lymphocytes are injected into the patient alone and in combination with the PSA epitope. The specific anti-tumor and PSA-specific immune response to the treatment is determined by methods comparable to those described in Example 34.

Similar human clinical trials may be conducted for treatment of patients with other TAA-expressing cancers, by replacement of the gene encoding CEA with a gene encoding another TAA into the recombinant vector of the present invention.

Please delete the paragraph on page 102, lines 26-30, and replace it with the following paragraph:

CAP-1 (Tsang, K.Y. et al, J. Natl Cancer Inst. 87(13):982-990, 1995), CEA amino acid position 571-579 YLSGANLNL **(SEQ ID NO: 23)**, CAP1-6D (Zaremba, S. et al, Cancer Res. 57(20):4570-4577, 1997) YLSGADLNL **(SEQ ID NO: 24)** and Flu peptide, influenza matrix protein peptide 58-66 GILGFVFTL **(SEQ ID NO: 42)** greater than 96% pure, were made by Multiple Peptide System (San Diego, CA).

Please delete the paragraph on page 105, lines 11-22, and replace it with the following paragraph:

A modification of the method described by Scheibenbogen et al (Scheibenbogen, C. et al, Clin Cancer Res 3(2):221-226, 1997) was used to measure IFNγ production to determine CAP-1 specific T cells. Briefly, 96-well Milliliter HA plates (Millipore Corporation, Bedford, MA) were coated with 100 μl of capture antibody against human IFNγ at a concentration of 10 μg/ml. After 24 hours incubation at room temperature, plates were blocked for 30 min with RPMI-1640 containing 10% human pool AB serum. 1×10^5 cells to be assayed were added to each well. CAP-1-6D-pulsed C1R-A2 cells were added into each well as APC at an effector:APC ratio of 1:3. Unpulsed C1R-A2 cells were used as negative control. HLA-A2 binding Flu Matrix peptide 58-66 (GILGFVFTL) **(SEQ ID NO: 42)** were also used as control. The responding cells were determined by the use of a Domino Image Analyzer (Otpomax, Hollis, NH).